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Gastroscopy and Esophagoscopy

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The official Publication of The American Gastroscopic Society

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The Bibliography should conform to that used by the Cumulative Index: e.g., King, J. M. & Woelfel, G. F.: Submucous Lipoma of the Stomach, Am. J. Surg. 83:32-34 (Jan.), 1952.

Illustrations: Six illustrations per article are allowed without cost to the author. Color illustrations, if desired, must be at the expense of the author.

THE PRESIDENT'S COLUMN

C. W. Wirts, M.D.

The prime objectives of most medical organizations are twofold; first, to hold scientific meetings which provide a suitable environment for the exchange of ideas on subjects of mutual interest to the membership and, second, to record and publish their proceedings so that these may be studied at leisure and those who have been unable to attend the meeting may have an opportunity of reading about it.

Because our Society is relatively young and small the efforts put forth to gain these objectives may very well be more arduous than is the case in an older organization with less specialized limitations. This year marks a renewed effort in the life of the Society to develop a publication which will both meet our needs and still be within the sphere of our capabilities. I am sure we all appreciate the recent combined efforts of the editor and the new editorial board in bringing out one of the best issues of the Bulletin so far. That there is still room for improvement we all agree. We also agree that this is a team effort and that any changes that are undertaken must be supported by all of us if they are to prove effective. The major portion of the meeting of the Board of Governors in Chicago on November 7th was devoted to ways and means of accomplishing this.

The editor and editorial board have now been authorized to obtain the services of a paid editorial assistant and further, to make provision for authors to review their galley proofs and obtain reprints. Additional changes will undoubtedly have to be anticipated and it is hoped that the membership at large will send in their written recommendations as well as be prepared to discuss the problem at the New Orleans meeting.

Because of the popular demand indicated by the postcard poll conducted by our secretary, Arthur Olsen, gastroscopic photography (including colored moving pictures), biopsy and cytologic diagnosis of gastric and esophageal disease, and the management of esophageal disease will form the three broad symposia for our annual meeting on the afternoon of Wednesday March 30, 1960. An excellent program is planned with particular attention being given to audience participation. I sincerely hope you will all make an effort to come and join in the proceedings.

The Management of Gastric Polyps

Travis Bridwell, M.D. and Robert J. Priest, M.D.*

Gastric polyps pose a problem of management and a guiding philosophy is needed to solve this problem. Is it mandatory that all gastric polyps be removed surgically, or can criteria be established to permit medical followup? We have reviewed our experience in an attempt to provide criteria for a rational decision in the management of these lesions. The diagnosis of 'polyps' has been made for any benign-appearing tumor of the stomach seen in roentgen study or by gastroscopic observation. Polypoid lesions were excluded if they were obviously of malignant appearance. However, the precise criteria necessitating surgical removal of gastric polyps is difficult to determine by a review of the medical literature [1, 2, 3, 4, 5]. The choice between partial gastrectomy or a limited excision is difficult to evaluate unless complete confidence can be established in the diagnostic differentiation of benign from malignant lesions. A further question to be solved is whether polyps considered benign may later undergo a malignant degeneration. Are there clinical and diagnostic criteria in which confidence can be placed for a safe medical followup? The following survey was done in the attempt to find an answer to these questions.

Incidence

We have reviewed over 3,000 gastroscopies done in the past 20 years. Seventy-four patients were found to have gastric polyps by gastroscopic examination, an incidence of 2.4 per cent. Yarnis et al [6] found an incidence of 1.6 per cent in 2,500 gastroscopies. Six additional patients were included in our study who were diagnosed by roentgen examination but had not been gastroscoped. Forty-three of the 80 patients were males and 37 were females. There were 6 negroes in the group, all of whom were females. The youngest patient was 23 years of age and the oldest was 85, the average age being 55.7 years. Table 1 shows the age and sex grouping of these patients. Approximately half of the cases fell between ages 55 and 70. Carey and Hay [7] found that over half of their series of 71 patients with gastric polyps were over 60 years of age. Hardt, Steigmann, and Milles [3] reported that 70 per cent of their patients belonged to the sixth and seventh decades, while Wheelock, Atkinson, and Jennings [4] found gastric polyps most commonly in the fourth and fifth decades of life.

Gastroscopic Findings

Seventy-four patients were gastroscoped. Single polyps were found in 30 patients and multiple polyps were seen in 31 patients. In Table 2 are listed the polyps diagnosed by gastroscopy. Single and multiple polyps have been found to occur with equal frequency in several reports [6, 8, 9]. Carey and Hay [7] found multiple polyps in only 39 per cent of 71 patients gastroscoped. Eighteen of our patients had a total of 26 progress gastroscopies. In 3 patients polyps seen previously were not visualized at the second examination. One patient had 6 progress gastroscopic examinations at yearly intervals without change in his polyp. Three other patients had significant changes in the progress gastroscopic observations. In 1 patient a polyp had increased in size, after 1 year, but remained unchanged 2 years thereafter. In a second patient 3 new polyps were found 1 year later with bleeding. These were found to be benign after they were excised. An area of stiffening of the mucosa with bleeding was seen in a third patient by progress gastroscopy 3 months after the initial examination. An operation to remove the gastric polyp and the suspicious area was advised but refused by the patient. He has been followed 4 years thereafter and remains in good health.

The location of the polyps in our series included 18 in the prepyloric area, 15 along the lesser curvature, 44 in the fundus of the stomach, 2 in the cardia, and 12 patients with polyps in 2 or more of these areas. Previous reports have indicated that polyps appear most commonly in the

lower third of the stomach [6, 9]. There were 10 patients in whom polyps were not seen gastroscopically but were diagnosed by radiographic examination of the stomach. All of the 8 malignant lesions were visualized through the gastroscope.

Table 3 lists the gastroscopic appearance of the gastric mucosa. The patients with malignancy included 2 patients with normal gastric mucosca, 3 patients with superficial gastritis, and 2 patients with atrophy of the gastric mucosa. Malignant polyps have not been seen in association with hypertrophic gastritis [1, 9]. None of the malignant polyps in our series were associated with hypertrophic gastritis. However, 8 of our patients with benign gastric polyps were considered to have a hypertrophic gastric mucosa. Yarnis et al [6] reported that they had not seen a true gastric adenoma with hypertrophic gastritis, although Carey and Hay [7] considered 9 of the 71 patients in their series to have benign polyps with hypertrophic gastritis.

X-ray Findings

All 80 patients in this series had roentgen examination of the stomach. Table 4 is a tabulation of the roentgen findings. In 22 cases (27 per cent) the x-ray examination failed to demonstrate a polyp or polyps which were seen later by gastroscopy. In the series reported by Carey and Hay [7] roentgen diagnosis failed to show 54 per cent of the gastric polyps and in the study by Hardt et al [3] 32 per cent of gastric polyps were not diagnosed by x-ray technique. Progress x-ray studies were done on 24 patients. There was a time interval of 1 month to 11 years between progress x-rays.

In a group of 8 patients with gastroscopically demonstrated polyps, not shown on x-ray examination, only 1 was found on repeat roentgenogram. There was no significant change in the roentgen appearance of gastric polyps in progress studies of the remaining 16 patients. Carey and Hay [7] obtained progress x-ray studies on 20 patients with benign polyps, none of whom had a change of diagnosis on repeat examination.

Laboratory Findings

Seventy-eight patients had hemoglobin determinations and the average value was 13.1 grams per cent. Twenty patients, 18 of whom were females, had a hemoglobin level below 12 gms. Of 40 patients tested for occult blood in the stool, 20 gave positive results. Only 1 patient had hematemesis. Five patients were seen with massive upper gastro-intestinal hemorrhages. One patient was treated medically because of advanced age (85 years) although she had hemorrhages on 5 occasions.

Gastric analysis was done on 70 patients. Achlorhydria was present in 30 patients, of whom all but 2 were stimulated by histamine injection. The average fasting free hydrochloric acid was 27 degrees, the averag maximum free hydrochloric acid was 34 degrees. Nine patients had acid curves over 50 degrees. Four of the 8 patients with malignant lesions of the stomach had a histamine-refractory achlorhydria. Miller, Eliason, and Wright [2] in reporting 8 cases of malignant degeneration in gastric polyps, found that all 8 cases were achlorhydric. Yarnis el al [6] reported achlorhydria in 91 per cent of their cases of gastric polyps. Hardt, Steigmann, and Milles [3] found that 75 per cent of their patient had no free hydrochloric acid. Five patients with malignancy had anacidity in the series reported by Carey and Hay [7]. Two patients in our group had pernicious anemia. One patient developed pernicious anemia 4 years following a patrial gastrectomy for benign polyps. There had been a histamine refractory achlorhydria before the operation. The second patient with pernicious anemia had been followed by annual stomach x-ray studies for 2 years before multiple gastric polyps were diagnosed by roentgenologic and gastroscopic techniques.

Table 5 lists the pathologic findings in 39 patients who had gastric surgery for polyps. There were 8 malignant lesions in this group, or approximately 20 per cent. Six of the malignancies were adenocarcinoma. One patient had an adenocarcinoma and an associated benign polyp. One of the most interesting surgical specimens contained 3 benign polyps and a fourth polyp with an

intraepithelial carcinoma (fig 1). This patient had a partial gastrectomy without delay because of the suspicious factors of achlorhydria and anemia, with gastroscopic and roentgenologic confirmation of the multiple polyps.

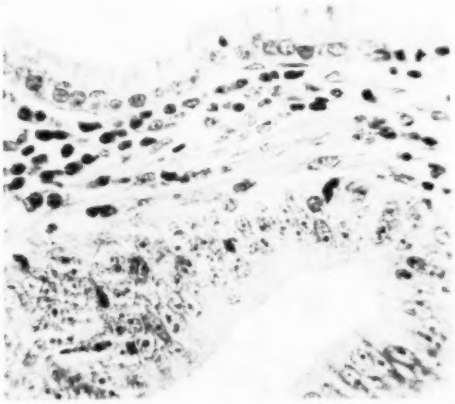

Figure 1. Intramucosal carcinoma as illustrated in the lower gland of a gastric polyp. Three other polyps resected at the same time were benign. (H. & E.: x 480)

Case Report

M.C., # 267455, a 63 year old white housewife complained of epigastric pain of 7 weeks' duration. Two gastric polyps were demonstrated by roentgen examination of the stomach (fig. 2). Three benign appear-

Figure 2. Roentgenogram of the stomach with two radiolucencies representing gastric polyps.

ing polyps were seen by gastroscopic observation. The largest of the polyps was 1 cm. in diameter. The stools were negative for occult blood. There was gastric achlorhydria after histamine stimulation. The patient had been under observation for arteriosclerotic heart disease, obesity and arterial hypertension. Angina pectoris was a prominent symptom. It seemed advisable to follow the gastric polyps with progress gastroscopy and x-ray studies, which were unchanged after 9 months.

The patent was hospitalized 18 months later with a myocardial infarct, and further progress gastroscopies seemed contraindicated. The gastric polyps were seen in a progress x-ray of the stomach 1 year later. However, there was an increase in the size of the polyps and the possibility of malignant change was considered. The polyps were excised from the stomach wall following gastrotomy, and all 3 polyps were benign on histologic examination.

Six months following the gastrotomy a 1 cm. gastric polyp was seen on x-ray examination. The polyp, on a stalk, was located on the lesser curvature of the proximal antrum (fig. 3). Because of the pa-

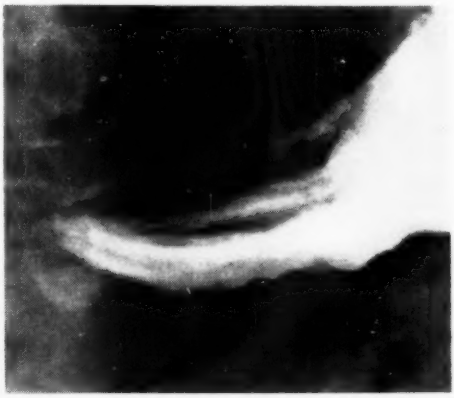

Figure. 3. Radiolucency representing a polyp on a stalk appearing in the stomach of the patient in fig. 2 six months following the local resection of three benign polyps.

tient's cardiac complication further surgery was avoided. Progress roentgenograms at 6 month intervals demonstrated no change in the radiographic appearance of the polyp.

Comment: This patient illustrates the dilemma of decision for medical or surgical management of gastric polyps when cardiac or other systemic disease adds to the difficulty of the decision. Local resection of the polyps was performed because of their increase in size and the associated gastric achlorhydria. However, a fourth and presumably new polyp was demonstrated in progress x-ray studies 6 months after the gastrotomy.

Factors Influencing Management

Thirty-eight patients were followed without surgical intervention and 39 or nearly 50 per cent underwent surgery. Two patients refused surgery. One patient died before an appropriate decision for management of the polyps had been determined.

Twenty-eight per cent of Carey and Hay's [7] 71 patients, 37 per cent of Hardt's [3] 62 patients, and 37 of Yarnis, Marshak and Friendman's [6] 73 patients underwent surgery.

Table 6 lists the various surgical operations of the 39 patients. Partial gastrectomy was done in one-half of the cases and 16 of the 39 patients had a local excision of the gastric polyp. Thirty-two of the 39 patients were operated on within 1 month after a decision for surgical management had been determined. Four patients were referred for operations after progress x-ray or gastroscopic examinations led to the suspicion of malignancy associated with the gastric polyps.

In attempting to determine the reason for medical or surgical management, each case record was studied carefully and an effort was made to select the factors that seemed most directly related to the final decision. Factors considered were the initial x-ray, progress x-rays, initial gastroscopy, progress gastroscopies, achlorhydria, hemorrhage, and age. Several unpredictable factors also influenced the decision. For example, 1 patient had an apparent epigastric mass, but a benign polyp was removed by excision. Peptic ulcers influenced the decision for surgical management in 2 patients.

In Table 7 are tabulated the factors which seemed to influence the ultimate mode of treatment. Initial gastroscopy was the most frequent decisive factor for followup without surgical intervention. In 14 of the 41 patients recommended for operation the initial x-ray and the initial gastroscopy provided the basis for decision. In this category were included 4 of 8 malignant polyps. The combination of achlorhydria and hemorrhage was the indication for surgery in another patient with a malignant polyp. The eighth patient with a malignant lesion was operated upon because of the findings in the initial x-ray and progress gastroscopy 1 month later.

Discussion

The association of adenocarcinoma and benign gastric polyps, either single or multiple in the same stomach, has been reported by several writers [6, 10, 11]. One case was encountered in this study. The development of a malignancy in stomachs where benign gastric polyps have been removed has been mentioned by several authors [1, 3, 8, 10]. To our knowledge no one has reported previously the development of a polyp in the stomach following surgery for gastric carcinoma. Such a sequence occurred 8 years after subtotal gastrectomy in 1 of our patients. In another instance a polyp appeared in the stomach remnant 12 years following a subtotal gastrectomy. Operation in the latter case had been advised because a malignancy had been suspected. Partial gastrectomy has been widely employed in this country for a relatively short period. A 15-year survival following surgery for carcinoma of the stomach has been reported recently [14]. There is a large group of patients available today who have had partial gastrectomy. We feel these patients are potential polyp formers and should have periodic progress roentgen studies.

Klein and Geller [11] have reported an interesting case where a carcinoma was found in the same area that a benign appearing gastric polyp had been visualized with the gastroscope 2 years previously. Yarnis et al [6] report a similar experience. The interval in the latter case was 4 years. Three possibilities were sugested by Yarnis. First, that an adenoma had undergone malignant transformation; second, that it was a carcinoma from the very beginning; and third, that an independent carcinoma completely overgrew and destroyed an original adenoma. The gradual development of a benign gastric polyp into carcinoma has not been observed clinically.[7, 9, 12] Carey and Hay [10] state that many gastric polyps have been followed for years and have not been seen to become malignant. During 13 years' gastroscopic followup they had not seen malignant degeneration in benign gastric polyps. The fear of possible malignant degeneration is the disturbing problem in the natural history of benign gastric polyps. The circumstantial evidence for

malignant degeneration of benign gastric polyps has been reported numerous times and we believe this to be the source of confusion. No one has actually observed clinically this transformation over a period of time. Fibiger [16] was able to feed nematodes to rats with the development of gastric polyps which subsequently showed malignant degeneration with distant metastases. Meulengracht [15] stated that half or more of all intestinal polyps degenerate but that malignant degeneration is rare in the stomach. Kiefer and Christiansen [12] believe that gastric polyps, like other intestinal polyps, can degenerate and must be considered precancerous lesions.

The reported incidence of malignant degeneration of gastric polyps varies considerably. Brunn and Pearl [5] reported an incidence of 12 per cent. An incidence of 35 per cent of 23 cases has been reported by Miller et al [2]. Paul and Logan [9], from reviewing the literature, cited incidences from 6 to 51 per cent. In our study only 1 case of intraepithelial carcinoma in a polyp was encountered. We believe malignant degeneration can occur but the exact incidence in gastric polyps is difficult to determine precisely.

Gastroscopy has been used widely as a diagnostic technique in this country for only 20 years. It became apparent to us from our review of these case reports that greater accuracy had been attained in our gastroscopic diagnosis in recent years. For example, 3 of the 8 malignancies in this study were polypoid adenocarcinomas which had not been differentiated from gastric polyps when gastroscopic experience at our institution had been limited. Gastroscopy has become very important in the evaluation of gastric polyps and in the subsequent decision for medical or surgical management.

A review of the literature indicates a lack of agreement on the management of gastric polyps. A polyp appearing malignant, a polyp prolapsing into the duodenum causing obstructive symptoms, or a gastric polyp that bleeds usually requires excision. The decision for management is more difficult for the asymptomatic gastric polyp which appears benign by x-ray or gastroscopic examinations. The excision of all gastric polyps has been recommended [1, 2, 3, 4]. Overgaard [8] believes that a gastric polyp indicates a pathologic condition which may develop into carcinoma, but is not itself a precancerous lesion requiring radical treatment. Hay [13] suggested non-operative management and a continuous followup for asymptomatic patients with benign appearing polyps less than 2 cm. in diameter. He advised partial gastrectomy for polyps over 2 cm., smaller polyps appearing malignant, patients with symptoms referable to gastric polyps, and patients who refuse or cannot accept adequate observation. Yarnis et al [7] believe that conservative management with frequent gastroscopic observations should be reserved only for the small solitary adenoma presenting benign characteristics.

In our experience we have not found that size or number of polyps, gastric acidity, and gastroscopic appearance of the gastric mucosa have been reliable criteria for the initial decision for management. We believe that progress x-ray and gastroscopic examinations are necessary to make a final decision for management in some patients. Gastric polyps that are benign in appearance can be followed with reasonable safety if progress roentgenologic and gastroscopic examinations are done at suitable intervals.

Summary

Eighty patients with gastric polyps have been studied from a group of 3,000 gastroscopies (2.4 per cent). The factors influencing the decision for management have been evaluated, including initial and progress x-ray and gastroscopic examinations and other clinical and laboratory criteria. Small gastric polyps, benign in appearance, can be followed by progress x-ray and gastroscopic observation. One polyp with an intraepithelial carcinoma was encountered and one patient with adenocarcinoma had an associated benign polyp. It would appear from our experience that partial gastrectomy has been done more frequently than necessary. Gastrotomy with local excision of benign appearing gastric polyps is adequate treatment when a decision has been made for surgical management rather than a medical followup.

TABLE 1

Age and Sex Incidence

Age Group	*Female*	*Male*	*Total*
20-29	0	3	3
30-39	4	1	5
40-49	8	9	17
50-59	6	11	17
60-69	13	18	31
70-79	5	1	6
80-89	1	0	1
	—	—	—
	37	43	80

TABLE 2

Gastroscopic Findings

Single polyps	30
Multiple polyps (2 or more)	31
Polyps not seen	11
Polypoid malignancy	2
Patients not gastroscoped	5
Unsuccessful gastroscopy	1
TOTAL	80

TABLE 3

Gastroscopic appearance of gastric mucosa

Normal Mucosa	32
Atrophic gastric mucosa	17
Superficial gastritis	14
Hypertrophic gastritis	8
Post-operative gastritis	1
Erosive gastritis	1
Infiltrative neoplasm	1
TOTAL	74

TABLE 4

Roengenologic Findings

Polyps seen	46
Polyp not seen	24
Stiff antrum	5
(4 benign; 1 malignant)	
Carcinoma	3
Giant rugal folds	2
TOTAL	80

TABLE 5

Pathologic findings of operated cases

Solitary benign polyp	11
(1 with associated gastric ulcer)	
Multiple benign polyps	12
(1 with associated duodenal lipoma)	
Heterotopic pancreatic tissue	2
Adenocarcinoma	8
(7 with associated benign polyp)	
(1 with intraepithelial carcinoma of polyp)	
Normal stomach	2
Miscellaneous	3
(giant rugal folds, marginal ulcer and multiple ulcers)	
TOTAL	38

TABLE 6

Surgical procedures in operated cases

Local excision	16
Partial gastrectomy	20
Total gastrectomy	2
Gastrotomy	1
(Polyp not found)	
TOTAL	39

TABLE 7

Factors determining management

	Medical	*Surgical*
Initial x-ray and gastroscopy	5	14 * (4)
Initial gastroscopy	10	3
Progress gastroscopy	6	3
Initial x-ray	2	8 * (2)
Progress x-ray	3	2
Progress x-ray and gastroscopy	5	1
Miscellaneous (age, achlorhydria, hemorrhage, mass, associated findings, etc.)	8	10 * (2)
TOTAL	39	41

* *Malignancy*

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DISCUSSION

DR. JAMES B. CAREY (Minneapolis, Minn.): The suggestion I shall make to resolve this controversy is to call the various things that we see through the gastroscope by their right names. Instead of calling all of the tumorous elevations that we see in the gastric mucosa, polyps, let us call some of them benign adenomas, some of them adenocarcinomas, some of them multiple polyposis, some of them carcinoma villosum, and some of them hyperplasia with atrophic mucosa.

I think if we would do that we would not get into arguments as to what to do about them, because if you make up your mind that a growth is a benign adenoma, you will leave it alone; if you make up your mind that something is an adenocarcinoma, you will advise its removal. Multiple polyposis, a general condition of the mucosa, is an entirely different matter, and the other insignificant adenomas and small masses of hyperplasia occasionally seen in atrophic gastric mucosa, particularly with pernicious anemia, are also different from adenomas or adenocarcinomas.

So, if we would be willing to abandon the general term polyp, which, after all, only postpones our decision, and come to a decision on these matters, confusion would be avoided.

Now, coming to a decision means, as Dr. Priest pointed out, in many cases repeated examinations.

The instance that he mentioned of the very early adenocarciomatous change is similar to an experience of my own. One polyp in a group of four changed its appearance and I changed my opinion about it, but three of them were still benign adenomas. But I believe that the one I subsequently called adenocarcinoma had always been an adenocarcinoma. I have never seen growth that I called a benign adenoma degenerate, or deteriorate, or develop, or grow into an adenocarcinoma.

DR. CHARLES A. FLOOD (New York, N.Y.): I wonder why Dr. Priest feels that the size of the polypoid lesion may be an important factor in deciding what to do with it or how to classify it.

I ask this question for two reasons: one is because now it seems fairly clear that the polypoid lesions in the large bowel, under 1 cm. in diameter, are seldom found to be malignant if removed surgically, and it seems as though the literature contains very few small gastric polyps, that is, gastric polyps of less than 1 cm. in diameter, which have been shown to be malignant.

The other reason for directing this question is that many of the little polypoid lesions which are seen gastroscopically, if removed—those too small to be seen by x-ray—are found not to be adenomata of the stomach but simply are classified by the pathologist are rare hyperplasias of no logical significance.

DR. PRIEST: Of course, Hay recommended partial gastrectomy for all polyps over 2 cm. in size, and I am sure that size would influence our initial decision, particularly whether malignant or benign. However, in three instances in this series of 74 polyps, they were observed to increase in size and yet were benign when excised,

so size in itself, and increase in size in our series have not given us any precise criteria for benignancy vs. malignancy.

PRESIDENT BERK: Wasn't the gastroscopic diagnosis benign in the one case and the pathological diagnosis was unequivocally carcinoma?

DR. PRIEST: Yes. The carcinoma was resected simply because there were four polyps and one was bleeding. None were presumed to be, but one polyp was proved to be a malignant lesion.

(Question by Dr. J. Frederick Monaghan, Bryn Mawr, Pa.): How many benign polyps have you ever seen bleed in the stomach? The fact that a polyp was bleeding, wouldn't that make it suspicious, I have never seen these multiple polyps bleed.

DR. PRIEST: We had no patient with generalized polyposis. We referred to solitary polyps and multiple polyps in the stomach.

Not infrequently when the polyps are followed by progress gastroscopy, at a subsequent examination they will be seen to bleed.

---

* *From the Division of Gastroenterology, Henry Ford Hospital, Detroit, Michigan.*

Presented at the annual meeting of the Am. Gastroscopic Soc., June 7, 1959.

NOTES FROM THE INSTRUMENT MAKERS

As you will read in the Cameron ad elsewhere in this issue, Mr. Edwin Bicknell at Metro-Tec, and the Cameron Instrument Company have combined their talents and efforts in design and manufacturing of gastroscopic and esophagoscopic instruments. In a personal interview with Mr. N. H. Wallen, President of Cameron and Mr. Edwin Bicknell, Director of the Metro-Tec instrument department, great enthusiasm was noted and they expect better research and production from this new arrangement.

Mr. Frederick J. Wallace of A.C.M.I. reports that work is going along steadily in producing his examining and operating gastroscopes as well as esophagoscopes. Refinements have been accomplished in these instruments. Mr. Wallace, always interested in research and development, declares a new and fundamentally different development activity is being worked on. However, he says that the time is premature for a detailed report.

Mr. Louis Streifeneder, of the Eder Instrument Company, reports that he is very busy taking care of orders and readapting scopes for the new visual head. This visual head has been described in the advertising in this and other issues of the Bulletin.

In all, our instrument companies are hard at work in the development of facilities for better endoscopic examination of the esophagus and stomach. The photographic end has not been neglected, and you will note many fine accomplishments in this field. Others are to come in the future.

ROY L. SEXTON, M. D.
Editor

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Clinical Manifestations of Extensive Atrophy of the Gastric Mucosa (Advanced Atrophic Gastritis)*

Angelo E. Dagradi, M.D., Rachael K. Guth, M.D., and Stephen J. Stempien, M.D.

Atrophy of the gastric mucosa is not a normal anatomical accompaniment of aging (1) (2). When this condition exists, it represents a definite organic disease (3) and has serious morbid potentialities.

The gastric mucosa may be compared to the epithelial lining of a large gland and, as is true of other glandular organs with large reserves, extensive destruction of the epithelium usually must occure before there develops an interference with its function. The clinical methods presently in use are not sufficiently sensitive to detect abnormalities of function of the gastric mucosa unless a very large part of it has been destroyed.

Atrophy of the gastric mucosa may eventuate from the destructive action of ingested corrosive poisons (4) or radiation burn (21); as a consequence of repeated inflammatory insults of an idopathic nature (chronic superficial gastritis); or in association with certain metabolic and nutritional disorders (pernicious anemia, sprue, pellagra, hepatic cirrhosis (18), etc. With respect to this latter group it must be said that there is no evidence available to prove that glandular atrophy may develop without preceding chronic inflammation (4) (13-15). Intestinal metaplasia of the gastric mucosa, therefore, represents the end-state of an antecedent inflammatory disease, and the terms "gastric mucosal atrophy" and "atrophic gastritis" are interchangeable. When the atrophy has become sufficiently extensive, the resultant destruction of parietal cells results in anacidity (5) (17) (21).

Whether or not atrophy of the gastric mucosa is a symptom-producing disease has long been a subject for debate (4) (6-10). Possibly one of the main reasons for this difference of opinion devolves from the fact that most instances of this disease occur in the older age group—a time when other pathological states have often developed in the abdominal viscera to which we are more prone to ascribe symptoms. This study was made in an effort to clarify this clinical controversy and to delineate the more commonly encountered clinical conditions associated with gastric mucosal atrophy.

Methods and Material

We have selected from our gastroscopic case material all patients demonstrating advanced and severe gastric mucosal atrophy, atrophy which was extensive enough to be reflected in an inability of the mucosa to secrete detectable amounts of hydrochloric acid in response to histamine stimulation on one or more occasions, and in whom the clinical and laboratory findings could not be logically ascribed to any other cause. In order to satisfy these criteria, many patients demonstrating gastroscopic evidence of severe gastric mucosal atrophy were eliminated from this study—either because of the presence of varying amounts of free hydrochloric acid on gastric analysis or because of the presence of other associated intra-abdominal disease states.

All patients were carefully investigated, including routine laboratory tests, gastric analysis, liver function tests, sigmoidoscopy, and stool examinations. All underwent roentgenographic examination of the upper gastrointestinal tract, while gall bladder series, barium enema studies, and liver biopsy procedures were accomplished when indicated by symptoms referrable to these organs. Gastroscopy was accomplished in an effort to explain the gastrointestinal complaints of these patients or, as in the case of those patients not having gastrointestional complaints, in an effort to explain the presence of histamine-fast achlorhydria.

The gastroscopic diagnosis of astrophy of the gastric mucosa was confirmed in a small number of our cases by means of peroral biopsy specimens of the gastric mucosa. It was not deemed essential to obtain microscopic confirmation in all cases since the gastroscopic features were so clear-cut and characteristic. While it must be

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admitted that not all cases of gastric mucosal atrophy can be diagnosed by gastroscopy, when the gastroscopic estimation of degree of involvement is "severe" histopathologic examination of an adequate biopsy specimen will invariably confirm the diagnosis.

Analysis of Cases

This study comprises a total of 44 cases ranging in age from 30 to 89 years with the majority (64%) falling in the 50-69 year age group. The number in each decade from the third through the eighth were respectively 2, 6, 14, 14, 6 and 2. All patients were males; 13 had pernicious anemia; 16 were chronic alcoholics (includes 5 of whom had pernicious anemia).

In 40 cases (91%) the presenting complaint at the time of hospitalization was referable to the gastrointestinal tract, while in 4 the chief complaint was directed to other organ systems. Interestingly enough, all of these latter patients had pernicious anemia: in 2 the chief complaint was neurological in nature, in one fatigue referable to a severe anemia was the main complaint, while in the last case shoulder pain due to cervical discogenic disease was the chief difficulty.

Of the 40 patients having primarily gastrointestinal symptomatology, in 25 the symptoms were limited to the upper gastrointestinal tract, 2 were suggestive of disturbance of the lower gastrointestinal tract, while in 13 symptoms were suggestive of both upper and lower gastrointestinal tract disturbance. Of this latter group, in 3 the symptoms were primarily upper gastrointestinal in nature, in 4 primarily lower, and in 6 the symptoms were equally distributed.

The symptoms varied in duration. In the main, they were described to be of a chronic, recurring nature. In 9 cases symptoms were of less than one year's duration, in 9 cases they varied from one to 5 years in duration, in 18 cases from 5 to 40 years; while the duration of symptoms in 4 cases was not available in the medical record.

Table I

Gastrointestinal Complaints of Patients Having Severe Gastric Mucosal Atrophy

	Pure Cases of Atrophy	*Alcoholics*	*Alcoholics and P.A.*	*P.A.*	*Total*
NONE				4	4
UPPER GASTROINTESTINAL	(20)	(11)	(5)	(8)	(44)
a) Epigastric Pain or Distress	11	8	1	5	25
Mid-Abdominal Pain or Distress	1				1
"Burning"	5			2	7
Food-Alkali Relief	5	4		3	12
Aggravated by Food	3	1			4
Food Intolerance (Fats, spices)	4			1	5
b) Epigastric Tenderness	2			1	3
c) Anorexia, Nausea, Vomiting	9	6	2	3	20
d) Abdominal Bloating & Belching	5			1	6
e) Upper G.I. Bleeding	6	6		2	14
LOWER GASTROINTESTINAL					
a) Cramping Lower Abdominal Pain	3	1	1		5
b) Diarrhea	3	2	2	3	10
c) Constipation	3			2	5
OTHER					
a) Weight Loss	9	5		1	15
b) Weakness, Lassitude, Fatiguability	4	4	1	1	10

Gastrointestinal Symptoms

Abdominal pain was complained of by 31 patients (70.4%); in 25 patients it was located in the epigastric region, in 5 it was lower abdominal, and in one it was located in the mid-abdomen. The pain was described to be "burning" in quality by 7 patients in whom it was epigastric in location, and in 3 epigastric tenderness was present on physical examination. Those patients in whom the pain was lower abdominal in location, described it as being of a "cramping" or "colicky" nature.

Twelve patients experiencing upper abdominal distress described an "ulcer-like" syndrome with food-alkali relief; 5 of these patients complained of intolerance to highly-seasoned or "greasy" foods. In 4 patients the pain was occasioned or aggravated by food ingestion. In none of the patients in this latter group was there a complicating active superficial gastritis present—all demonstrating simple atrophy at the time of gastroscopic examination.

Gastrointestinal bleeding was described in the anamnesis of 14 of our 44 patients (32%). Six of the patients in this group were chronic alcoholics but had no evidence of varices, and 2 had pernicious anemia. Liver function tests were all normal in the alcoholic patients with one exception—a BSP retention of 14.5% in one case. Nine described melena alone, 3 hematemesis alone, while 2 experienced both hematemesis and melena. Three patients in this group were admitted for gross bleeding severe enough to require transfusions. Two of these entered the hospital because of hematemesis and one because of melena. In the remaining 11 the hemoglobin level, RBC count and hematocrit levels at the time of hospitalization were above the range necessitating transfusions. Seven described only one episode of melena while 4 described 2 or more such occurrences.

Twenty patients of the total group offered the complaints of *anorexia, nausea and vomiting*, alone or in combination. Three were patients with pernicious anemia, 6 were chronic alcoholics, 2 had both pernicious anemia and alcoholism, and 9 were pure cases of gastric mucosal atrophy. These symptoms, too, were intermittent in nature and periodic in occurrence. One patient was a ruminator—a habit he had almost his entire life-time.

Weakness, lassitude and fatigue alone or in combination were described by 10 patients. In 7 cases no immediate cause other than the gastric mucosal atrophy was ascertained to explain these symptoms, while in 3, anemia (1 due to pernicious anemia and 2 due to upper gastrointestinal bleeding) was the underlying explanation.

Abdominal bloating and belching was complained of by 6 patients in our group.

Symptoms referable to lower bowel disturbance were present in 15 patients—10 (22% of the total group) complained of *diarrhea* and five of *constipation*. Of these patients complaining of diarrhea, 3 had pernicious anemia, 2 were alcoholics, 2 were alcoholics with pernicious anemia, and 3 were pure cases of gastric mucosal atrophy. Frequency of bowel movements varied from 4-5 to 10-12 daily. The stools were soft or watery in consistency and contained no blood or mucus. Associated "colicky" lower abdominal pain was described by most of these patients. The diarrhea was episodic in occurrence and was of 2 to 7 years' duration. In all instances the diarrhea subsided promptly during hospitalization, and it was felt, although not proved, that the administration of dilute hydrochloric acid was an important factor in the improvement of these patients. In 2 patients of this group, diarrhea was the presenting complaint at the time of admission. Five patients complained of constipation. In 3 patients it was of life-long duration, in one it had been present for 3 years, and in another for 2 months (pernicious anemia).

Nutritional Deficiency

Weight loss was a presenting complaint in 15 cases. Five patients had lost 10-20 pounds during the six months preceding hospitalization, while 7 patients had lost 16 to 60 pounds in weight during a period of from one to 20 years preceding admission. In 3 cases neither the amount nor the duration of time during which weight loss had occurred was available in the hospital record. In 5 patients of this group

the weight loss was unassociated with the previously mentioned anorexia, nausea or vomiting.

Clinical evidence of avitaminosis was apparent in 9 patients—3 of whom had pernicious anemia and 2 of whom were alcoholics. Six complained of sore and burning tongue, which appeared reddened on inspection, and 3 had cheilitis.

Hematologic Manifestations

Eighteen patients were anemic. Secondary anemia was present in 5 and was attributed to blood loss. Primary pernicious anemia was present in 13 patients.

Neurologic Manifestations

Subacute combined sclerosis was present in 5 of the 13 patients having pernicious anemia. Peripheral neuritis was noted in 4 patients—2 of whom had pernicious anemia and 2 of whom were chronic alcoholics.

Neoplastic Changes

Gastric mucosal polyps were found in 2 cases. Carcinoma of the stomach developed in 2 other patients on follow-up observation: one was a patient with pernicious anemia and the other was a non pernicious anemia patient.

Discussion

Opinions regarding the symptomatic propensities of gastric mucosal atrophy range from those stating that the condition produces no symptoms to those claiming that it produces important symptoms. In general it may be said that the subjective symptoms of advanced chronic atrophic gastritis are not sufficiently characteristic to permit the diagnosis to be made from the case history alone. It is appreciated that a study of this type merely correlates symptoms with a finding and is not amenable to more objective scientific evaluation than this.

In order to reduce the extraneous factors to a minimum our selection of cases was limited to those having severe gastric mucosal atrophy on gastroscopic examination and histamine-anacidity on gastric analysis. All cases in which other clinical or laboratory findings could be interpreted to be responsible for the symptomatology were eliminated. There remained, therefore, three groups—patients with pernicious anemia, chronic alcoholics, and those having advanced atrophic gastritis alone (Fig. 1). It is significant that within each group the same spectrum of symtomatology was encountered to the same degree. We are left, then, with the fact that a certain constellation of symptoms occur in association with advanced gastric mucosal atrophy.

Ninety-one per cent of the patients in our study had gastrointestinal symptoms; the commonest of these being epigastric pain or distress which was present in 62% of the symptomatic patients. Interestingly enough, despite the achlorhydria, epigastric burning pain or distress was complained of in 7 cases, and an "ulcer-like" syndrome with food-alkali relief in 12. Anorexia, nausea, and vomiting occurred in 45% of the cases, while 34% had suffered appreciable weight loss. Bleeding from the gastrointestinal tract was noted to have occurred on one or more occasions by 14 patients (32%). It is impossible to be exactly certain of the mechanism for this but it may be postulated that the bleeding could have stemmed from the atrophic mucosa (hemorrhagic atrophic gastritis). It is not unusual to see frank hemorrhages and hemorrhagic erosions in such a gastric mucosa on gastroscopic examination (3).

Diarrhea was the commonest of the lower bowel complaints, occurring in 10 (23%) cases. Four of these patients were chronic alcoholics while 6 were not. One might speculate that the alcoholism per se might have been responsible for the diarrhea. However, it is also possible that the diarrhea was gastrogenous in its etiology and was due to the gastric anacidity. In all cases, the diarrhea came under rapid control during hospitalization and it is possible that the administration of dilute hydrochloric acid may have been the responsible factor.

In regard to the relationship of gastric mucosal atrophy to other disease states, it is to be noted that two of our patients had gastric mucosal polyps, and two others eventually developed carcinoma of the stomach. Although the relationship of

atrophy of the gastric mucosa to the development of gastric carcinoma is not entirely clear, it is not unusual to elicit a history of longstanding chronic dyspepsia in many patients having carcinoma of the stomach, and atrophy of the gastric mucosa is frequently present in association with gastric carcinoma.

Whether or not an acquired atrophy of the gastric mucosa may produce Addisonian pernicious anemia is a moot point. One may reason by analogy that the potentiality for this development is present since the end results (loss of intrinsic factor with resulting nutritional deficiency) are the same, whether the specific gastric mucosal elements are destroyed by progressive inflammatory disease, or by surgery as in total gastrectomy, or are absent due to a constitutional defect (juvenile pernicious anemia). The observations that anacidity may precede the onset of pernicious anemia by 4 to 25 years (11), that severe gastric mucosal atrophy may be present for years before clinical pernicious anemia develops (20), the development of pernicious anemia subsequent to an acute gastritis or a chronic gastritis (11-13), or total gastrectomy (22), the parallelism between average age of onset of pernicious anemia (16) and average age of patients having advanced gastric mucosal atrophy, the frequently elicited long history of gastrointestinal symptoms in patients with untreated pernicious anemia as described in the early writings on this disease entity (19), the possibility that the spontaneous relapses and remissions in the course of untreated pernicious anemia may be due to relapses and remissions of a chronic superficial gastritis affecting quantitatively the production of intrinsic factor (active superficial gastritis is frequently found in association with atrophic gastritis in patients with pernicious anemia), seems to favor the proposition that in some patients at least pernicious anemia is acquired, secondary to gastric mucosal atrophy.

Summary

1) A selected group of 44 patients showing severe gastric mucosal atrophy on gastroscopic examination, and histamine anacidity, and having no laboratory or clinical evidence of other intra-abdominal disease, was evaluated.

2) Forty patients (91%) of this group had gastrointestinal symptoms of varying nature, duration, and degree of severity.

3) The commonest symptoms associated with extensive gastric mucosal atrophy (advanced atrophic gastritis) are: epigastric distress; anorexia, nausea, vomiting; upper gastrointestinal bleeding; weight loss; weakness, lassitude and fatigue, and diarrhoea.

4) Equally important clinical manifestations associated with this disease were: pernicious anemia, secondary anemia, avitaminoses, neurological disease, gastric polyps, and gastric carcinoma.

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Discussion

QUESTION: Wouldn't it be necessary to gastroscope a number of patients without symptoms, to demonstrate that atrophy is not found in those patients, before you arrive at these conclusions?

DR. DAGRADI: In our study there were four patients who were gastroscoped because of the fact that they had achlorhydria. They had no symptoms related to the gastro-intestinal tract. Forty others were gastroscoped because of gastro-intestinal symptoms. People with gastric mucosal atrophy do not necessarily have to have symptoms. This does not mean that the condition is asymptomatic. This holds true for other disease states. For instance, people with gallstones do not necessarily have symptoms.

DR. LEONIDAS H. BERRY (Chicago, Ill.): If there is no one else who wishes to discuss this, I should like to say this is something of a pet subject of mine. I followed a group of sixty-some patients for a period of eight years, who had chronic atrophic gastritis. I am glad we have had one clinical paper today because I think unless we take into consideration clinical disease, we are just going through so much academic exercise, and I think that when we have such widely varied opinions as we heard this morning about the all-importance of biopsy—being 2 mm. of the gastric mucosa—and the diagnosis of gastritis based entirely on that, we ought to issue a word of warning not to go too far afield in throwing aside all of the widely accepted information we have had during the last twenty years about the importance of the gross picture in the diagnosis of gastritis.

I should like to confirm the clinical observations of Dr. Dagradi and associates and state that in a large number of people with gastric atrophy I have been able to elicit the same kind of symptoms, including a wide variety of manifestations which the speaker states is attributable to gastritis. However, instead of saying they are attributable to gastritis, I would say they are associated with gastritis.

I have treated a number of such patients with Vitamin B complex, liver extract, and improved diet with a striking improvement of symptoms and the gastroscopic picture after several weeks. This seems to indicate that the whole thing is probably a general disturbance of metabolism and dietary deficiency and what we see is only local manifestation of the general disease.

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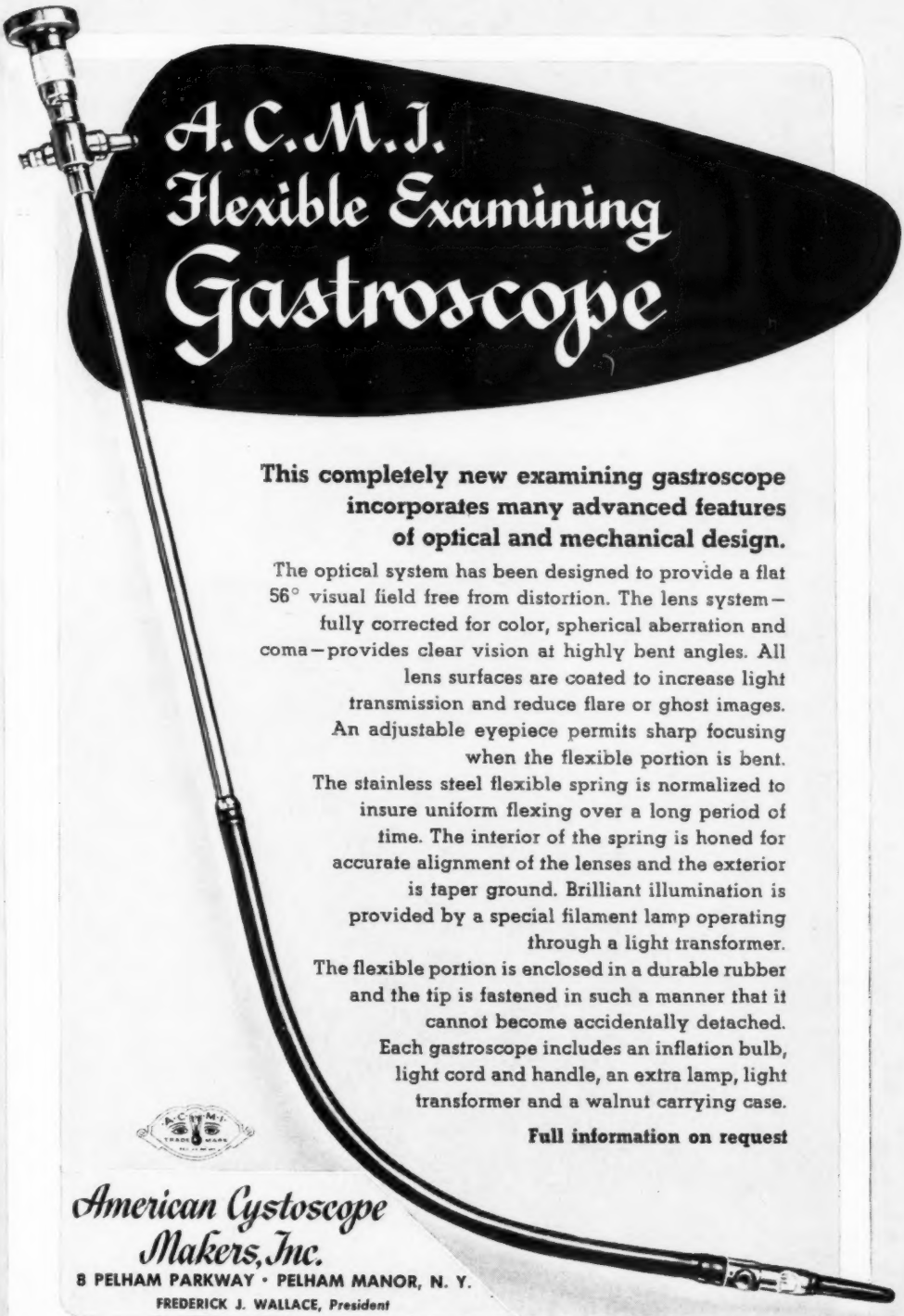
* *Presented at the Annual meeting of the American Gastroscopic Society, June 7, 1959. From the Gastroenterology Section of the Medical Service, Veterans Administration Hospital, Long Beach, Calif.*



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Gastroscopic Diagnosis of Chronic Gastritis

N. Henning, M.D. *

The gastroscopic diagnosis of chronic gastritis is a macroscopic diagnosis. It must be verified by histologic examination of extensive biopsy material. Only suction biopsy has proved suitable for routine use. Dr. Heinkel and I have modified this method. Up to the middle of February, 1958, we had studied 2,541 specimens from 2,100 patients, without a single complication in performing these biopsies. This paper concerns a comparison of the gastroscopic with the histologic diagnosis. In addition it is to demonstrate what can be accomplished with cytologic diagnosis. The cell-swab method which was described in 1949 was employed to obtain the material for the cytologic examination.

Before we take up the main subject, I shall demonstrate our biopsy findings.

Histologically we differentiate between the normal mucosa, superficial inflammation, and atrophic gastritis. These forms gradually merge into one another.

Normal mucous membranes contain pits which are uniformly dense. They are neither deepened nor dilated. The superficial epithelium is arranged in tall columns with uniformly large, chromatin-rich basal nuclei and with transparent cytoplasm. The interstitial tissue is hardly recognizable in the region of the specific glandular tissue. No increase in cells is to be found in the lamina propria and there is no predominance of plasma cells or of leukocytes. In the layer of the specific glands we recognize the chief (adelomorphous) cells, the covering (delemorphous) cells and the secondary cells.

To this normal histologic picture of the gastric mucosa there corresponds a characteristic cytologic picture (gastrocytogram). The smear preparation is deficient in cells. One finds mainly normal cylindric cells, that is, high columnar, uniform elements, with basal oval nuclei, and basal tail-shaped extensions of the cytoplasm. I shall later return to a special form of gastrocytogram in the presence of a hyperfunctioning mucosa.

In superficial gastritis, there are changes in the covering epithelium and in the Lamina propria mucosae, whereas the specific glandular tissue is not involved.

Chiefly in the region of the apices of the ridges, the covering epithelium shows unclear outlines and turbidity of cytoplasm. Some of the nuclei depart from their basal position. Anisokaryosis and anisochromia develop. In serious cases degenerative changes appear, such as karyorrhexis and karyolysis. Papillary lengthening and flattening of cell groups may alternate. The small pits vary in distribution (density) and when there are changes in the lemina propria mucosae they are deeper and serpentine.

Widening and proliferation of cells is typical of the lamina propria mucosae, plasma cells accounting for most of the cellular elements. This cellular infiltration is limited to the lamina propria mucosae, the glandular parenchyma remaining free. A more dense infiltration with leukocytes, combined with leukopedesis is regarded as an acute exacerbation of the inflammation.

There are no findings in the gastrocytogram that are characteristic for superficial gastritis, because we examine only the covering epithelium. As a rule cytologic studies disclose changes in the covering epithelial cells. They are short cylindrical to cuboid, and have a relatively large roundish nucleus, which at times may contain large nucleoli. Intraepithelial leukocytes are indicative of leukopedesis and of an acute exacerbation. These cytologic characteristics may be interpreted as signs of gastritis, without suggesting anything about the histologic form of the gastritis.

Atrophic gastritis is histologically characterized by changes in all zones of the mucosa. The changes in the covering epithelium may be more pronounced than in case of superficial gastritis. The lamina

* *Director University of Erlangen, Medical Clinic, Erlangen, Germany.* *Read before The American Gastroscopic Society, seventeenth annual meeting, Washington, D. C., May 24, 1958.*

propria mucosae is wider and shows cellular infiltration. The small pits are serpentine and penetrate to the muscularis mucosae. The most typical sign is the reduction in the specific glandular parenchyma that may progress to complete atrophy. The preserved cell groups may be completely dedifferentiated, chief cells and covering cells can no longer be distinguished. An increase in the interstitial connective tissue divides the remnants of the specific glands into separate lobules. Especially characteristic for the late stages is the transformation of the surface epithelium into goblet cells, i.e., goblet cell metaplasia with loosening of the cell-deficient lamina propria mucosae.

The gastrocytogram of atrophic gastritis is characterized by a cuboid or indifferent surface epithelium within noticeable anisocytosis, anisokaryosis and severe plasmic vacuolization. Atrophic gastritis is proved by the presence of goblet cells, which are readily identified by their club-like distension, a large vacuole and a flat basal nucleus.

The greatest changes in the exfoliated cells are found in the atrophic mucosa that characterizes pernicious anemia. I mention the great enlargement of the cell nuclei with shifting of nucleus/cytoplasm ratio, anisokaryosis, a tendency to multiple nuclei, enlarged nucleoli, and a predominantly peri-nuclear arrangement of the formation of vacuoles in the cytoplasm. The so-called active pernicious cells with large, chromatin-rich nuclei and large nucleoli are, however, no definite proof of pernicious anemia; we have also found them in atrophic gastritis without signs of Addison-Biermer's anemia. These cellular changes are to be interpreted in their entirety as precancerous abnormalities suggesting that, from the cytologic viewpoint, atrophic gastritis appears to be a precancerous condition.

My explanations so far have described the biopsy and the cytologic findings, which monotonously are found again and again if we restrict our theme to diffuse chronic inflammation of the stomach. We now come to the main question: Is gastroscopy adequate for the detection of chronic gastritis? Or, expressed more exactly, does macroscopic-endoscopic observation of the mucosa permit definite statements regarding its histologic condition?

In the comparison that follows chronic gastritis, according to Schindler's classic nomenclature, is divided into superficial, atrophic, and hypertrophic gastritis.

After we had performed biopsies for some time on all patients with gastric symptoms, we began our analysis with the cases of duodenal ulcer. This was done because many of these patients had the same etiopathogenesis, the same syndrome of complaints, the same functional disturbance of the stomach in the form of increased motor activity and secretion, the same roentgenogram, and the same endoscopic picture. For decades we had seen, gastrocopically, with remarkable regularity a mirror-like mucosal surface, which was not coated. However, it showed, frequently only in the lower portions of the fundus, fine or coarse granulations, which we had interpreted earlier as signs of hypertrophic verrucous gastritis. If there was any relationship at all between clinical, roentgenologic, gastroscopic and histologic signs, then it should be particularly evident in the presence of duodenal ulcer.

I would like to mention some historically interesting findings in the gastrocytogram, the interpretation of which at first proved difficult. Since 1948 we have examined cells of the gastric mucosa which had been obtained with the cell-swab method; this was about three years before we had a properly functioning biopsy device. In the presence of duodenal ulcer with maximal diurnal and nocturnal gastric secretion, we found three types of cells which did not originate from the covering epithelim.

The first type of cell is large, polyhedral to spherical. The small, round nucleus is eccentrically located; two or three nuclei may be present. The acidophilic cytoplasm, stained orange-red by Pappenheim's method contains fine, luminous yellow granules, which are visible also by phase-contrast microscopy. These cells correspond to the covering cells of the mucosa of the fundic region.

A second type of cell is smaller, predominantly plump and cylindrical. The fairly large, round nucleus is located at one end of the cell. The pale cytoplasm is

filled with coarse basophilic granules, which, with Pappenheim's stain appear dark blue to black. These elements correspond to the chief cells of the mucous membrance of the fundic region.

Attached to the covering cells one finds additional smaller cylindrical cells with nongranular, readily flowing cytoplasm; we have regarded these as secondary cells.

At first we attempted to interpret the appearance of cells from the depth of the parenchyma of the fundic glands as a loosening of the cellular structure, as a result of the gastritis which we presumed to exist. Later, biopsy explained the peculiar process to us.

It is a matter of an increased exfoliation of the specific fundic cells, which appear in the form of cylinder-cells (casts) in the foveolae and which are taken up by the swab-probe. We see in this peculiar mechanism the morphologic manifestation of an increased cell catabolism in the presence of maximal hyperactivity of the glands of the fundus regon.

The histologic findings in duodenal ulcer early in our experience revealed with considerable regularity a completely normal mucosa. As the number of cases increases this regularity decreased, and our results are shown in Table I.

In superficial gastritis we found chiefly mild changes. Hypertrophic gastritis was not observed.

The special status of the mucosa in duodenal ulcer compared to other gastric disorders is most clearly demonstrated by a comparison with a gastric ulcer and carcinoma. Whereas normal mucosa was found in 44.6% of the cases of duodenal ulcer, the corresponding percentages in 204 cases of gastric ulcer was only 18.5% and in 83 cases of gastric carcinoma only 16%.

The Reliability of Gastroscopic Diagnosis in Atrophic Gastritis

On the basis of gastroscopic diagnosis, in 1935 we found an incidence of 8%, this was conformity with later results reported by Wiebenga. For the present biopsy studies 1,476 patients with stomach complaints but without ulcer or carcinoma were available. We found the histologic picture of atropric gastritis in 363 cases, or 24.5%. Thus atrophic gastritis is three times more frequent than could be assumed on the basis of the earlier gastroscopic findings.

Let us see how the gastric muscosa of patients with histologically proven atrophic gastritis appeared at gastroscopic examination. Conversely, we shall examine the results of histologic study in cases in which the diagnosis of atrophic gastritis was made gastroscopically.

Tables II and III show that only in about 25 of the total number of cases of established atrophic gastritis do the gastroscopic findings agree with the histologic findings; 75% of the cases are therefore missed by gastroscopy. If on the other hand, the diagnosis of atrophic gastritis is made by gastroscopy, it is confirmed histologically in about 50% of the cases. In this category one deals with more severe cases, particularly with those of Addison's anemia.

The Reliability of Gastroscopic Diagnosis in Superficial Gastritis

In a group of 1,476 patients with gastric complaints in the absence of ulcer and carcinoma, we found the histologic picture of superficial gastritis in 43.5%. This form of inflammation, therefore, occurs among our patients twice as often as atrophic gastritis.

The comparison with the gastroscopic diagnosis was carried out in the same way as was atrophic gastritis.

Table 4 shows that only 37% of the total number of cases of established superficial gastritis can be detected with the gastroscope, while in the predominant remainder of cases erroneous diagnoses are being made. If we analyze histologically only those cases in which the diagnosis of superficial gastritis has been made with the aid of the gastroscope, the corresponding histologic correlation is found in only 46%. (Table V.)

Thus, in endoscopically diagnosed superficial inflammation the diagnosis is confirmed histologically in only about 50% of the cases.

Before drawing conclusions from the results of our studies, I would like to ward

off an objection which might be made against our diagnoses made by biopsy. Are we justified in assuming that the entire mucosa is of the same character as that observed in a small piece of it? To clarify this question two or three biopsy specimens were obtained in each of 342 cases (a total of 708 specimens) from various areas of the fundus, and the histologic pictures were compared. Differences were observed in only 12 per cent of the cases.

The second biopsy specimen showed an abnormal picture in only 4 per cent of 71 cases with gastroscopically normal mucosae, and the abnormal aspect was always that of superficial gastritis. In 187 cases of superficial gastritis the incidence of a differing biopsy was 12 per cent. There we found in the control biopsy specimens either a normal mucosa or one of atrophic gastritis, but never both. A differing aspect was observed in 25 per cent of 103 cases of atrophic gastritis, consisting only in combinations with superficial gastritis. Among the 12 per cent of differing cases, therefore, we found combinations of normal mucosa with that of superficial gastritis and combinations of superficial gastritis with atrophic gastritis, but not combinations of normal mucosa with that of atrophic gastritis. Altogether we are justified in concluding from these results that the histologic aspect of a single biopsy specimen permits with a high degree of probability the inference of the same character for the entire mucosa of the fundus. On the other hand, the observed combinations of the histologic aspects in the rare instances in which two biopsies from the same stomach differed suggest that the chronic inflammatory process of the gastric mucosa takes a course from a stage with a normal aspect to that of superficial gastritis and from the latter to that of atrophic gastritis.

Our comparative studies have reference only to the fundic mucosa, for various reasons. The mucosa of the fundus is the proper field for gastroscopic diagnosis. Here we find the best conditions for endoscopic observations, the highest degree of brightness and the optimum distance of the mucosa from the lens, as well as the vertical projection. In the antrum there is less light, the distance is greater with disproportional reduction of the object, and the mucosa is seen at an angle, with shortening of the picture. On the other hand, the antral biopsy is much more difficult. A more elastic sound is required and it must be pushed close to the pylorus under roentgenologic control. The mucosa of the antrum is more tough and consequently greater demands are made on the biopsymeter. Finally, the interpretation of the histologic picture in the antrum is difficult, because there is little basis for clear distinction between normal and abnormal pictures. Nevertheless, we did not neglect the histologic study of the mucosa of the antrum, but this material will not be presented here.

The gastroscopic concept of hypertrophic gastritis does not find any correlation in the histologic picture. From the pathologic-histologic view point hypertrophic gastritis, therefore, does not exist, not even in association with gastric erosion which always shows inflammation in the erosive area. We were able to show that only three histologic aspects of the mucosa of the fundus were observed within the frame of our studies, and that they reappeared again and again with great monotony, namely, normal mucosa, superficial gastritis and atrophic gastritis. These three forms gradually merge into one another. We have reasons to assume that from the pathologic-histologic point of view there is only one inflammatory process, which finally terminates in atrophy. It follows from this statement that the many forms of gastritis which have been described by some gastroscopists do not exist. At this point I would like to call attention to the fact that Rudolf Schindler, the great pioneer in gastroscopic diagnosis, always abstained from fantastic interpretations.

In comparing the diagnoses of gastritis made by gastroscopy with these made by histologic examination, the superiority of the latter method is to be expected. The macroscopic examination of a mucosa must be inferior to the examination of a histologic section. One cannot, therefore, be surprised when the gastroscopic diagnosis

of gastritis is reliable only to a small degree. Except for the severe forms of atrophic and erosive gastritis, the exact diagnosis of chronic gastritis may be made only by histologic examination with the aid of a technique which is neither painful nor dangerous and which can be a routine method used regularly in all cases of gastric complaints. The cytologic examination of the exfoliated cells yields results more rapidly, but on the whole it is inferior to biopsy.

With the routine use of biopsy it becomes possible to confirm therapeutic effects objectively with the aid of a control biopsy.

Our studies compel us to conclude that in the future, for the sake of an exact diagnosis of gastritis, one should always attempt to make a histologic diagnosis in addition to a gastroscopic diagnosis.

TABLE 1

Histologic picture in duodenal ulcer

	Cases	%
Normal	78	44.6
Superficial Gastritis	85	48.6
Atrophic Gastritis	12	6.8
Total	175	100

TABLE 2

Gastroscopic diagnosis in histologically proven atrophic gastritis

	Cases	%
Normal Pattern	101	30.0
Superficial Gastritis	129	38.2
Atrophic Gastritis	84	24.8
Hypertrophic Gastritis	24	7.0
Total	338	100

TABLE 3

Histologic picture in gastroscopic diagnosis of atrophic gastritis

	Cases	%
Normal Structure	27	16.5
Superficial Gastritis	53	32.3
Atrophic Gastritis	84	51.2
Total	164	100

TABLE 4

Gastroscopic diagnosis in histologically proven superficial gastritis

	Cases	%
Normal Pattern	225	43.4
Superficial Gastritis	192	37.0
Atrophic Gastritis	53	10.2
Hypertrophic Gastritis	49	9.4
Total	519	100

TABLE 5

Histologic picture in gastroscopic diagnosis of superficial gastritis

	Cases	%
Normal	97	23.2
Superficial Gastritis	192	46.0
Atrophic Gastritis	129	30.8
Total	418	100

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Myo-Epithelial Hamartoma of the Stomach*

Robert S. Nelson, M. D.

Diagnosis of small submucosal tumors of the stomach may be made more frequently by gastroscopy than by roentgenological examination, if the method is used routinely to evaluate undiagnosed and frequently typical upper abdominal pain. The clinical value of such a diagnosis is apparent when simple wedge resection of the small, benign tumor results in complete pain relief for the patient. The following instance demonstrates this possibility, and is similar to several cases previously reported.[3]

Case Report

A 63-year-old colored male, employed as a mixer in a candy factory, was first seen April 18, 1958, complaining of epigastric pain. His discomfort occurred irregularly, but frequently, occasionally being accompanied by nausea without vomiting. There was no history of hematemesis, melena, or weight loss, and he uses no alcohol, tobacco or coffee. Physical examination was essentially normal throughout. Laboratory examinations included a normal urinalysis, a hemoglobin of 15.0 gm., WBC's 5,600, with a normal differential, and a negative serology. Results of liver functions tests, including serum proteins, bromsulfalein retention, serum billirubin, alkaline phosphatase, thymol turbidity, and cephalin flocculation were within normal limits. Three stool specimens were positive for occult blood, and negative on smear and culture for organisms and parasites. X-ray examination of the heart, lungs, gall-bladder, colon, esophagus, stomach and upper small intestine showed no abnormalities. Sigmoidoscopic examination was also within normal limits, but on gastroscopy there appeared to be moderate atrophic changes, and a small submucosal tumor of the lesser curvature of the stomach at the junction of the body and fundus was visualized. No ulcerations were noted. Repeat x-ray examination failed to visualize the tumor, but at gastroscopy it was again visualized at the same location (Fig. 1). Exploratory laparotomy was carried out May 2, 1958, and a submucosal tumor found and removed from the stomach at the described location. The patient did well following the operation except for a wound infection on the sixth postoperative day, which resulted in some separation of the abdominal muscles. The pathologic report on the specimen was as follows: 1) "Resected portion of stomach, chronic gastritis with focal acute erosions; 2) Hamartomatous nodule in gastric mucosa. Comment: There is no recognizable pancreatic tissue in the suspected mucosal nodule. There are, however, certain duct-like spaces lined by mucos-producing columnar epithelium. These may be hamartomatous or may simply be dilated gastric pits occasioned by scarring due to the inflammatory process." The final pathological diagnosis was hamartomatous nodule of the lesser curvature of the stomach (Fig. 2).

Figure 1. Gastroscopic Photography of Submucosal Tumor of Lesser Curvature.

The patient was followed in the clinic, and admitted 5 months later for repair of a ventral hernia. He stated that he had been entirely free of his previous epigastric pain during this interval, and was discharged following his repair in good condition.

Discussion

There has been considerable confusion in the terms used to designate these small submucosal tumors involving the stomach wall. While aberrant or heterotopic pancreas has been recognized since 1859, the term hamartoma was first used in 1904 to include any type of aberrant tissue in the

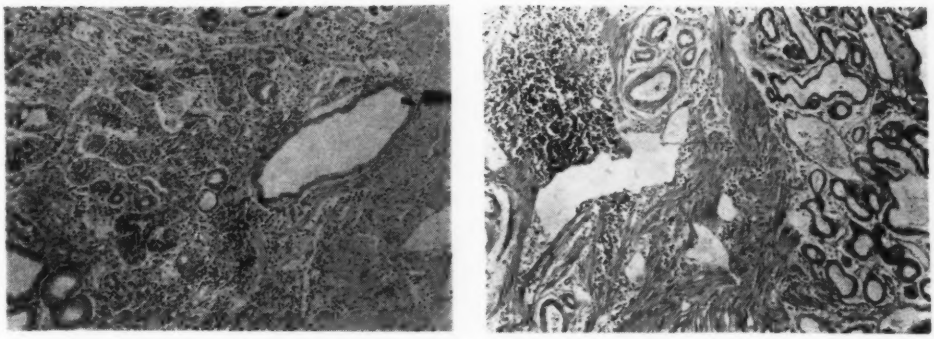

Figure 2. (a) and (b) showing microscopic structure of the submucosal tumor visualized on gastroscopy.

gastrointestinal tract, and "myo-epithelial hamartoma" was advocated in 1940,[1] to apply to all of these lesions of the gastrointestinal tract which appear to be due to aberrant tissue, whether pancreatic components were present or not. In one review of non-malignant tumors of the stomach in 1951,[4] no mention is made of myo-epithelial hamartoma, although there is a complete section on aberrant pancreas. However, this nomenclature appears to be widely accepted, and the terms heterotopic pancreas, aberrant pancreas and myo-epithelial hamartoma of the stomach used interchangeably.[2, 5, 7]

It is generally agreed that these small tumors are the result of embryologic rests, regardless of their degree of later differentiation. In the present case the tumor, when resected, was regarded as a hamartoma by the Pathology Department and it was suggested that the nodule might have been caused by chronic inflammatory changes in the stomach. In one review of epithelial heterotopia of the alimentary tract[6] it is noted that small nodules may occur in the edges of chronic ulcers. Our patient's stomach showed only superficial erosions, however, and the situation is not that found in chronic ulcerations. At surgery, the gross appearance was typical of heterotopic pancreas as seen in other portions of the stomach. The location was atypical, although heterotopic pancreas has been reported in all areas of the gastric mucosa. The term myo-epithelial hamartoma is used to designate the tumor because no definite pancreatic tissue was found on histological examination, and this may be a more general term. There can be little doubt, however, that the tumor represents an embryological rest and might well be called aberrant pancreas as suggested by other observers.

Summary

A case is presented of myo-epithelial hamartoma of the stomach occurring in an unusual location and visualized only by gastroscopic examination. The tumor was felt to represent aberrant pancreatic tissue in the stomach, although no definite pancreatic cells were found on histologic examination. Simple block resection of the lesion led to complete relief of symptoms.

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Leiomyoma of the Stomach with Hemorrhage Diagnosed Pre-Operatively by Gastroscopy and Roentgen Examination *

Julius G. Parker, M.D., Herman W. Lubetsky, M.D. and George R. Gerste, M.D.

Among the more uncommon causes of hemorrhage from the upper gastrointestinal tract are ulcerations of non-epithelial tumors. Palmer (1) in his review of the literature found 610 cases of benign gastric leiomyomas. Of the 140 patients in whose histories pertinent information was given, it was found that 42 per cent presented with gross gastric hemorrhage. Among 5,194 gastric tumors of all types, which Palmer gathered, 82 or 1.95 per cent were leiomyomas. It has been variously estimatel that about 50 or 60 per cent of benign non-epithelial gastric tumors are leiomyomas. Cole and Berry (7) estimated the incidence of gastric leiomyomas among benign gastric tumors to be about 37 per cent, Collins and Collins (8) quoted about 40 per cent, Eliason and Wright (9) found it to be 60 per cent.

The following is a case report of a leiomyoma of the stomach with ulceration and massive hemorrhage. The correct diagnosis of a mesothelial tumor with ulceration was made pre-operatively both by gastroscopy and barium-meal study.

Case Report

This 39-year-old, white female housewife (E.K.) was admitted to Montefiore Hospital of New York City, on 10-4-55 with a history of three tarry stools during the preceding three days. There was no vomiting, pain or diarrhea. In the previous six years, there had been vague digestive disturbances, but a barium-meal about six years prior to this admission was reported elsewhere as normal. In 1953, she was found to have hyperthyroidism and was treated medically with radio-active I^{131} Physical examination revealed a white female in no acute distress. She appeared pale, but there was no evidence of shock. Blood pressure was 120/65 and pulse rate was 100. No thyroid was palpable. Abdominal examination revealed no masses, tenderness or other abnormalities.

On rectal examination, a small amount of stool, slightly positive to hematest, was present. The remainder of the examination was normal. The initial laboratory examination, on 10-4-55, revealed a hematocrit of 23 per cent with 8,500 white cells and normal differential. The urinanalysis was normal. The B.U.N. was 23.5 mgs per cent and fasting blood sugar was 83 mgs per cent. A gastro-intestinal series, on 10-6-55, revealed a 3 x 4 cm filling defect on the greater curvature aspect of the stomach. This filling defect caused some medial displacement of normal mucosal folds which were somewhat stretched. In addition, there was in this filling defect a central collection of barium measuring about 1 cm in its greatest diameter. On the filled films, the central collection is surrounded by a large zone of radiolucency corresponding to the tumor mass in the wall of the stomach. See Figures I and II. The esophagus, duodenal bulb and duodenal loop were normal. On the four hour examination, there was a persistent collection of barium in the zone of ulceration within the stomach. The diagnosis was a benign leiomyoma of the corpus of the stomach, but a leiomyosarcoma could not be excluded. Gastroscopy was performed on 10-11-55. Local anesthesia was induced with 2 per cent Pontocaine. An Ewald tube was passed and nothing could be aspirated from the stomach. The gastroscope was passed with ease. There was a sessile polypoid tumor present on the greater curvature of the posterior wall of the mid corpus about 8 cms from the cardia. The tumor was about 4 cms in diameter and was covered by smooth, pale red mucosa, except at its summit where there was a deep ulcer crater measuring 1 cm in diameter. The rugal folds of the corpus stopped abruptly at the margins of the tumor. The remainder of the gastric folds were smooth and pale red in color. The antrum appeared normal. The gastroscopic diagnosis was a submucous mesothelial tumor, leiomyoma or leiomyosarcoma.

Figure 1. Barium filled study demonstrating the filling defect along the greater curvature aspect of the stomach with a central barium-filled niche.

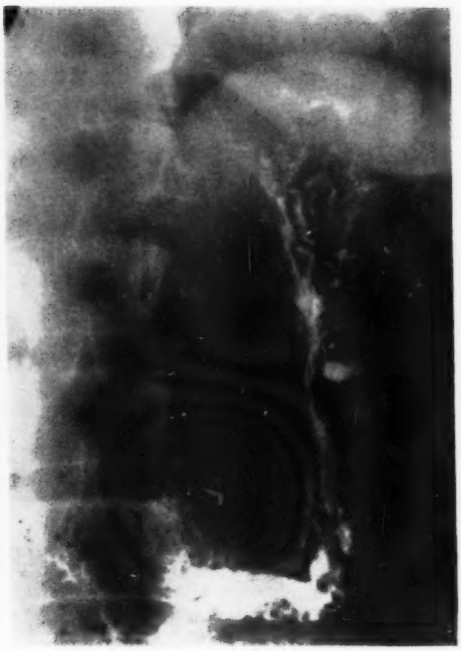

Figure 2. Mucosal study demonstrating the filling defect along the greater curvature aspect of the stomach with a central barium-filled niche.

Course in Hospital

The patient was given 1,500 cc of whole blood during the next twelve days. This raised her hematocrit to 35 per cent. There was no further bleeding. Surgery was performed on 10-21-55. A right paramedian incision was made. The rectus muscle was retracted laterally and the peritoneum opened. On exploration, a mass measuring 5 x 5 x 6 cms in size was noted on the posterior wall of the stomach near the greater curvature opposite the incisura. This mass was sharply delineated in the wall of the stomach. No metastases were seen. A subtotal gastrectomy of the Polya type was performed using an anticolic gastrojejunostomy. The abdominal wall was closed in layers.

Pathological Report of Resected Specimen

Grossly, the serosal surface of the resected portion of the stomach contained a 4 x 3.5 cms thickened, injected and firm area on the posterior wall adjacent to the greater curvature. The stomach was opened on the lesser curvature aspect. In the region corresponding to the firm area felt on the serosal surface was a 4 x 3 x 3 cms mass of dense yellowish-gray, focally whorled tissue which completely replaced normal musculature. This mass was covered by mucosa and serosa. The mucosa in the central portion of this mass was ulcerated by an area 1.5 cm in diameter. The ulcer penetrated for a distance of 1 cm into the tumor mass. The mucosa appeared normal elsewhere.

On microscopic examination, the tumor was covered by normal gastric mucosa except in the ulcerated area. There were focal areas of hemorrhage in the base. The tumor completely replaced the muscularis. The overlying serosa was fibrotic with infiltration by chronic inflammatory cells. The tumor was composed of interlacing bands of smooth muscle and fibrous tissue. There was no evidence of any malignant change. Final diagnosis: leiomyoma of stomach with ulceration.

The patient had an uneventful recovery and was discharged from the hospital 24 days after admission. She was last seen in February, 1959 and there has been no evidence of recurrence nor any complications due to her surgery. Follow-up studies of her gastro-intestinal tract by means of barium meals on 2-9-56 and 5-29-57 revealed a resected stomach with a normal functioning stomach remnant and gastroenterostomy. There is no evidence of recurrence of this lesion nor appearance of a new lesion.

Discussion

The gross appearance of a leiomyoma of the stomach has been described many times and is characteristic enough, both by x-ray (3) and gastroscopy to make a definitive diagnosis possible before surgery, as occurred in the instance being reported.

Palmer in his review uncovered 17 patients that had been studied gastroscopically. The tumor was visualized in all but one of the 17 patients. The observed tumors he reported varied in size from 1.5 to 20 cms in greatest diameter and 13 were in the sub-mucous position. The characteristic gastroscopic finding of a leiomyoma is as follows: The surface configuration is usually smooth and discrete in outline but may also be nodular or uneven. The mucosa over the tumor is usually of normal color and texture, but appears stretched and there is a loss of the usual rugal folds. When an ulceration is present, the mucosal folds can be followed to the margin of the ulcer. An important feature is that of the ruge mounting the sides of the tumor from the surrounding mucosa. The mucosa of the remainder of the stomach may appear normal or may appear atrophic. In two other cases collected by Palmer, peristalsis was found to pass without impairment. Schindler, Sandweiss and Mintz (4) and Da Gradi (5) have reported individual instances of gastroscopic identification of the leiomyoma proved subsequently at surgery.

The roentgen criteria for diagnosis of intra-mural tumors of the stomach have been previously stated by Elliot and Wilson (2) and Sahler and Hampton (6). The characteristic roentgen findings may be summarized as follows: 1) demonstration of a sharply circumscribed, smooth, intraluminal filling defect; 2) abrupt obliteration of gastric mucosa over the tumor with preservation of normal gastric pattern in the immediate surrounding area; 3) preservation of pliable gastric walls and peristaltic activity; and 4) demonstration of an ulceration in the central portion of the tumor in many cases. In contrast to an extrinsic pressure defect, the uninvolved wall of the stomach forms an abrupt angle with the tumor defect.

The therapy of an intra-mural tumor such as a leiomyoma is surgical removal. Medical therapy is contraindicated since ulceration with hemorrhage is so common and differentiation between a benign and malignant lesion cannot be made without histological examination.

Summary

1. A case report of a gastric leiomyoma with hemorrhage as the initial symptom is presented.

2. The lesion was diagnosed both by gastroscopy and roentgen examination preoperatively.

3. There have been no sequelae three and one half years after surgery.

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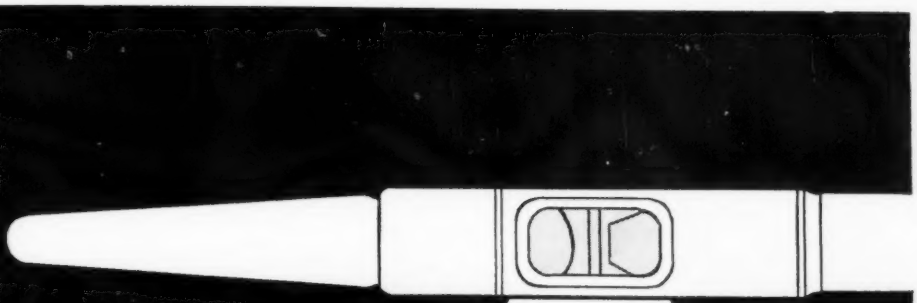

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